less or great, with the portion of the human anatomy that has more than all others to do with assimilation and metabolism.

As a student, it was assigned to me to demonstrate the shock of acapnia, described by Yandell Henderson. This experiment made a tremendous impression upon me, and today, as a result of this demonstration, to a large extent I take the utmost care in protecting the viscera from undue exposure. I recommend this to you in the fullest degree. The type of incision, the less retraction of the wound, the exposure from the abdominal cavity of only the area to be repaired, and the use of hot, moist tapes are the essentials.

Could one see each individual cell with the naked eye, what injury of tissue would be our vision. The avoidance of contusing and crushing clamps where possible, the clean knife-blade severance of tissues, the noninjury of blood supply, the careful hemostasis with fine, nonirritating suture material, and the careful approximation of tissue with nontraumatic needles and proper sized, absorbable suture means quicker healing, less absorption of broken-down proteose material, and therefore less shock.

One would not think of radical surgery in an acute or subacute, pelvic condition. One would not attempt any constructive surgery in the presence of an infectious lymphangitis. It has been demonstrated that the gastro-intestinal wall is pervious to a dye such as acriflavin. It has been observed that in a colitis, microörganisms invade the bowel wall and contiguous tissue. In inflammatory conditions, as a large, penetrating, or slowly perforating duodenal or gastric ulcer, or in an intestinal obstruction, be it acute or chronic, as from a malignant, rectal growth, there is a septic invasion of the bowel wall and neighboring tissue. The peritoneum will handle only a relative amount of infection, and the body functions of assimilation, metabolism, and elimination under such circumstances are already quite disturbed. Therefore it is our duty not to proceed with radical surgery in these inflamed, potentially infected areas, and expose wide, raw surfaces for septic absorption. We should first allow the tract to return to normal by some more temporary or multiple stage measure, as, for example, a Billroth II or Devine exclusion procedure for a complicated duodenal or gastric lesion; a cholecystostomy for an acutely infected gall bladder; or a preliminary enterostomy for a malignant obstructive lesion of the intestinal tract.

## CONCLUSIONS

1. In consideration of publications and recommendations let us always have a critical sense.

2. Before we proceed with surgery let us ask ourselves this question: Will this procedure aid the body processes to return to normal the most surely and the most completely?

3. Let us not be just skillful artisans, but let us be scientific surgeons and mindful of the importance of anatomy and pathology, let physiology guide our surgery of today and tomorrow.

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# CARDIOSPASM*

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DISCUSSION *by D. D. Comstock, M. D., Los Angeles; Kenneth S. Davis, M. D., Los Angeles.*

## GENERAL CONSIDERATIONS

THE esophagus is a neuromuscular tube about nine inches long. It connects the pharynx with the stomach and serves for the passage of food. Much of our conception of the physiology of deglutition we owe to Meltzer [1] and Cannon [2] and to their respective coworkers.

In the process of swallowing, a solid bolus is propelled by the wave of peristalsis, which is characterized by the simultaneous dilatation of the lower segment immediately in front of, and constriction behind the bolus. On arriving at the cardia the sphincter, which is ordinarily closed, relaxes for a moment to let the food pass into the stomach. On account of the difference in the musculature the rate of peristalsis is much faster in the upper part than in the lower. The innervation of the esophagus is derived from the vagus and sympathetic fibers—plexus of Auerbach and Meissner. Cannon has shown that in the lower part of the esophagus the waves of peristalsis travel independently of the vagus. Thus the sympathetic nerves of the lower esophagus and cardia are capable of the propulsion of food when called upon, regardless of the integrity of the extrinsic nerves.

One of the disorders of the lower esophagus in which dysphagia and obstruction play a prominent feature is cardiospasm. This is characterized by dilatation and hypertrophy of its musculature associated with functional stenosis of the lower end, usually of slow onset and long duration.

## HISTORICAL

The first authentic case of cardiospasm appears to have been reported by Purton [3] in 1821. In 1878 Zenker and von Ziemssen [4] collected seventeen cases in the literature. Little interest was aroused, however, until 1881, when von Mikulicz [5] recognized and described the etiological significance of spasm in diffuse dilatation of the esophagus. The original theory of Mikulicz placed the site of the disease at the cardia, and the spasm as the primary cause, hence he called this condition cardiospasm.

Rosenheim [6] attributed the cause to atony of the esophageal musculature.

Krause [7] suggested that simultaneous development of cardiospasm and paralysis of the circular

* A Critical Review of the Literature—With Analysis of Forty-Four Cases Treated at the Massachusetts General Hospital During the Past Fifteen Years.

Thesis submitted to the faculty of the Graduate School of Medicine, University of Pennsylvania, Philadelphia, in partial fulfillment of the requirement for the degree of Master of Medical Science.

The final year of graduate study under the extramural preceptorship of Harris P. Mosher, M. D., professor of laryngology, Harvard Medical School; chief of throat service, Massachusetts General Hospital and Massachusetts Charitable Eye and Ear Infirmary, Boston, Massachusetts.

muscle, due to degenerative change in the vagus, might be the etiology.

Einhorn [8] in 1888 considered that the obstruction might be due to the incoördination of the muscular movement of the esophagus.

Rolleston [9] in 1896 advanced a similar view, but more explicitly placed the fault on the longitudinal muscular fibers, in that they did not help to dilate the cardia at the moment when the esophagus was ready to propel food into the stomach.

Zenker and von Ziemssen [4] thought the condition was congenital in origin. Walton [10] entertained a similar view and stated that the incoördinate action, whether it was spasmodic or simply a failure to relax, must have been present at birth. He argued that a considerable number of cases have been reported in young children; that many adults, when care was taken in eliciting the history, dated their symptoms back to early childhood; that among those whose symptoms were of a comparatively short duration roentgenological and esophagoscopical examinations often revealed advanced pathological changes in the esophagus, all of which strongly pointed to a congenital origin.

Held and Gross [11] believed that (*a*) the vegetative nerve imbalance, due to inherited weakness; and (*b*) the involvement of the vegetative system due to an inborn status asthenicus or status thymicolymphaticus, play an important rôle in the etiology of this disease. They also named (*c*) the infections by toxic and metabolic agents; (*d*) the reflex irritability from the neighboring organs as kidney, liver, and gall bladder; the diseases of lungs, of stomach and intestine; or (*e*) local disorders within the gullet as factors in the production of this condition.

Through anatomical, clinical and roentgenological studies Doctor Mosher [12] has clearly demonstrated that inflammatory changes in the structures both above and below the diaphragm brought on this condition. He has not insisted, however, that this was the sole cause of the disorder. He feels that there is an element of stricture and backward twist at the lower end of the esophagus, and that these two causes often act together.

In recent years Hurst [13] and independently of him, Brown Kelly [14] pointed out that fibrosis of Auerbach's plexus at the lower end of the esophagus interfered with the normal neuromuscular mechanism. They compared this process to the heart block which is caused by the degenerative change in auriculoventricular bundle. As to the fundamental causes of this neurotrophic disturbance nothing definite is yet known.

## INCIDENCE

*Age.*—The ages vary. Jackson [15] treated an infant two days old in whom esophagoscopy was done, effecting a cure. Langmead [16] reported a case in which the typical symptoms occurred immediately after birth, and at eighteen months roentgenogram and esophagoscopy confirmed the original diagnosis.

The youngest patient treated at the Massachusetts General Hospital was a boy five months old, the oldest a man seventy-two years of age. From the hospital records there, the writer collected forty-four cases of cardiospasm which occurred between November 1911 and November 1926, a period covering fifteen years. Of this number about one-half were in the third and fourth decades.

*Sex.*—Exclusive of those cases of globus hystericus in which there was no characteristic change in the esophagus, and by reason of which could not properly be classed as true cases of cardiospasm, there were twenty-five males and nineteen females, or, roughly speaking, a ratio of 5 to 4. Vinson [17] collected a series of five hundred cases at the Mayo Clinic; of these, 294 were males and 206 females. The ratio of these figures corresponds closely to those collected at the Massachusetts General Hospital.

## SYMPTOMATOLOGY

In well-developed cases the symptom-complex is pathognomonic. Those cases that seek our aid are usually of long standing, always progressive since their beginning. The onset may be insidious or abrupt. Pain, dysphagia, and regurgitation are the three cardinal symptoms.

In the series of cases just mentioned, pain of more or less severity was recorded in eighteen. Earlier in the disease the pain was no more than a sense of discomfort, invariably intermittent, lasting only an hour or two, followed with a variable period of freedom. There was substernal pain in ten, and four of these described this as being sharp and lancinating. The remaining eight had epigastric pain which radiated at times to the back and especially to the left shoulder. These patients were not infrequently treated as having a mild form of indigestion or possible cholelithiasis.

In fourteen patients dysphagia was the prominent symptom. These people complained of food "sticking under the sternum" and took quantities of liquids to wash it down. Later each act of deglutition was accompanied by more or less obstruction as the food passed the cardia. The patients voluntarily selected a type of food which they could take with the least difficulty. As a rule solids caused more trouble and required considerable mastication. A few subsisted on liquids or semisolids only. The temperature of the food ingested had a definite influence on some. Usually extreme heat or cold was not well borne.

Finally there was another group of twelve, the characteristic manifestation being regurgitation. At first this was intermittent, only a mouthful or two being lost during or after a meal. With dilatation of the esophagus, however, regurgitation was delayed for hours or even days. In a few cases nocturnal regurgitation had been reported; this carried a danger of possible lung complication. The quantity of fluid regurgitated at times was more than the meal just taken, indicating stagnation of food from the previous meals or collection of saliva. Lactic acid, butyric acid, and other fermentative products were not infrequently in the regurgitated material.

When the obstruction persisted because of so little food reaching the stomach, the patients

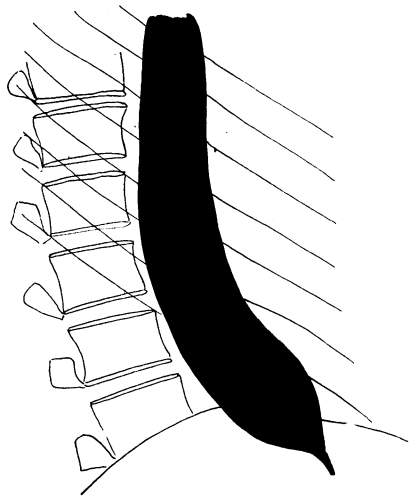

Fig. 1.—Spindle-shaped or fusiform esophagus.

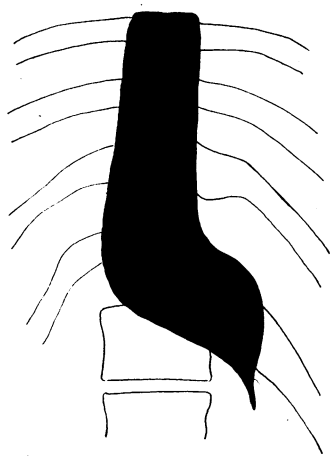

Fig. 2.—Pear-shaped or flask esophagus.

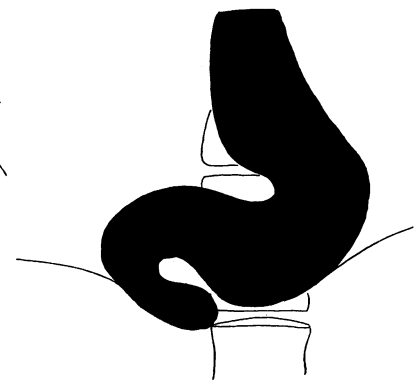

Fig. 3.—S-shaped esophagus.

steadily lost in weight. One of these cases was a woman who lost ninety pounds in five years, and another was down to sixty-eight and one-half pounds at the time of her admission.

PATHOLOGY

*Site of Stenosis.*—Originally Mikulicz laid the site of stenosis at the cardia. In recent years, however, this view has been much modified. The presence of the physiological sphincter has long been recognized.

Experimental work on rabbits by Payne and Poulton [18] has clearly shown that the part of the esophagus that plays sphincteric action extends some little distance above the diaphragm. Hurst [13] regards the last two or three inches of the human esophagus comprising the whole of the abdominal portion and sometimes to a very small part of the thoracic portion, as the sphincter. He bases this view upon the observation made during laparotomies performed for the relief of cardiospasm and a study of the esophagus in stillborn infants. Brown Kelly [14] holds a similar view, and it is at this portion of the esophagus that many other English observers place the site of "achalasia."

Jackson,[19] on the other hand, denies the presence of a sphincter at the lower end of the esophagus. He locates the cardiospasm at the "hiatus esophageus," and the spasmodic constrictions are of specialized muscle fibers there encircling the esophagus—the "diaphragmatic pinchcock."

Roentgenologic, esophagoscopic, and anatomic studies extending over a period of years led Mosher [12] to believe that the site of stricture is at what he terms the "liver tunnel." The terminal portion of the esophagus is not only surrounded by the cone of the diaphragm, but it is also encased by the left lobe of the liver. Owing to the position of the liver in front and the stronger left crus of the diaphragm behind, the terminal portion of the esophagus consists of a vertical part and a horizontal part. Where the two meet, the esophagus twists on itself and turns to the left.

In fluoroscopic studies on normal persons during the act of deglutition, when the diaphragm is up, the esophagus comes to a nipple-like point in the middle of the liver tunnel. With inspiration, when the liver is pressed down together with the diaphragm, then immediately the esophageal content is emptied into the stomach. In a certain number of cases of cardiospasm, Mosher pointed out, there is what he calls "reverse phenomena" in which the esophagus is closed momentarily when the liver is down, and opens when the liver is up. He considers that both the upper edge of the liver tunnel and the area at the junction of the two arms of the terminal portion of the esophagus are the usual sites of stenosis.

*Dilatation.*—Lambert [20] described three types of dilatation:

(*a*) The fusiform or spindle-shaped esophagus which is the commonest form of dilatation. It is characterized by the gradual widening of the lumen about midway between the cricoid cartilage above and the cardia below, where it gradually tapers to its normal size (Fig. 1).

(*b*) The pear-shaped or flask esophagus, the type on which Mikulicz laid special stress, is one in which the lower one-third or one-half of the esophagus is so dilated that it may at times be capable of holding two pints or more (Fig 2).

(*c*) The S-shaped is the rarest type. The two ends of the esophagus being fixed, with the increase in length, the esophagus bends until it becomes S-shaped (Fig. 3).

*Hypertrophy.*—Microscopically all the layers are thickened. The epithelium is generally thinned out. Later, as the fluids accumulate and fermentation goes on, the superficial layers are eroded and at some areas the entire epithelium is denuded. The mucosa is definitely thickened with evidence of chronic inflammatory changes as shown by the preponderance of plasma cells, small lymphocytes, and a few scattered polymorphonuclear leukocytes. The muscularis mucosa is also thickened. There is considerable fibrosis in the submucosa. Here the blood vessels increase in number as well as in

Fig. 4.—Longitudinal section of the esophagus at the lower end in a case of Doctor Mosher's. Male, 50, white; fifteen years' duration, finally died of pulmonary tuberculosis. The thickened muscularis mucosae shown in the center. The chronic inflammatory cells invading the deeper structures. Note the prominence of the blood vessels. Photograph by the writer.

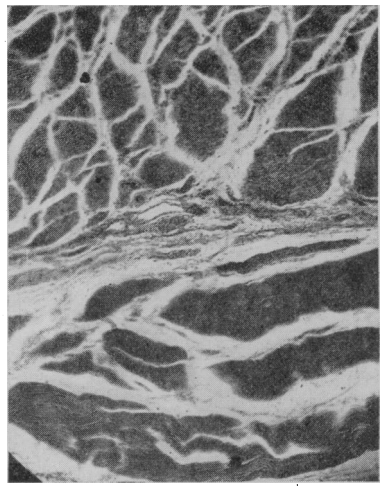

Fig. 5.—Hypertrophic muscular coats and the plexus of Auerbach without any ganglion cells. According to Dr. G. W. MacGregor, who prepared this tissue section, the average thickness of the inner coat measured 1.4 mm., the outer coat 1.1 mm., and the muscularis mucosae .3 mm. At the upper end of the esophagus one ganglion cell to four sections, and no cells in one hundred sections at the lower end. Photograph by the writer.

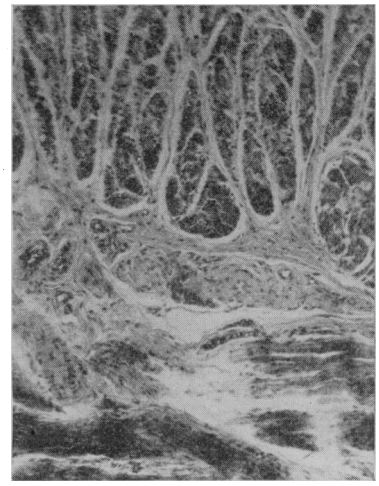

Fig. 6.—Longitudinal section of the lower end of esophagus of child at birth. The ganglion cells in Auerbach's plexus shown in the center. Photograph by the writer.

size, with marked perivascular infiltration by the inflammatory cells (Fig 4).

Both muscular layers are greatly hypertrophied (Fig. 5).

Associated with these changes there is perceptible diminution of ganglion cells in Auerbach's plexus. This is particularly conspicuous at the lower end and over the area where the dilatation is greatest (Figs. 6, 7, and 8).

Recently Irwin Moore [21] collected a series of pathologic specimens and published an excellent paper dealing with this phase in the study of cardiospasm.

## DIAGNOSIS

The diagnosis is rarely difficult. If care is taken in eliciting the history it seldom leads to a wrong conclusion. The pain may simulate gastric ulcer or gall-stone colic. Syphilis and the previous swallowing of caustic must be excluded. Benign stricture at the cardia is rare.

*Fluoroscopic Technique.*—Fluoroscopic examination is the best means at our command. At Doctor Mosher's clinic the following procedure is usually carried out: The patient is stripped above the waist to the skin and a sleeveless gown is placed over his head. He stands behind a fluoroscopic screen, facing the examiners—the roentgenologist, the surgeon and his assistants. Rapidly but carefully the patient's heart, lung fields, and the movement of the diaphragm are noted; then the attention is focussed on the esophagus and the fluid level is noted if present. A sufficient quantity of bismuth-milk mixture, usually about one-half glassful, is given by mouth and the contour of the esophagus is studied from the front and from the side. The opaque meal reveals the presence or absence of abnormal peristalsis, of hyperirritability, of hernia and diverticulum. As the fluid accumulates at the lower end, special attention is paid to the relation of the emptying time to the excursion of the diaphragm, the position of the lung tips and all other features that might assist in the diagnosis. Doctor Mosher's olive-tipped bismuth-coated rubber bag is indispensable in this work. It is much like the original Sippy bag and an inflating tube is connected to the ordinary Tyco sphygmometer as an indicator of the pressure exerted within. Rarely the pressure of more than five or six pounds is required. After the bag is withdrawn another glassful of the opaque meal, to which is added a dose of Seidlitz powder, is given. In a mild form of stenosis the

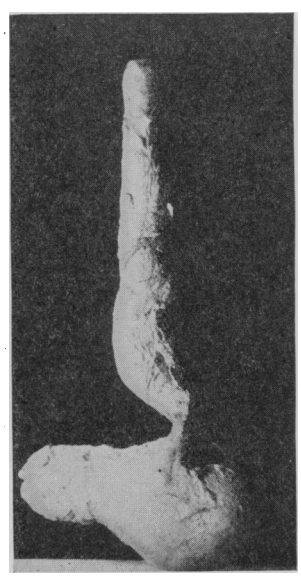

Fig. 7.—The cast of esophagus and stomach in case of cardiospasm. The esophagus moderately dilated at the lower half. From Doctor Mosher's laboratory, Harvard Medical School. Photograph by Werner Mueller.

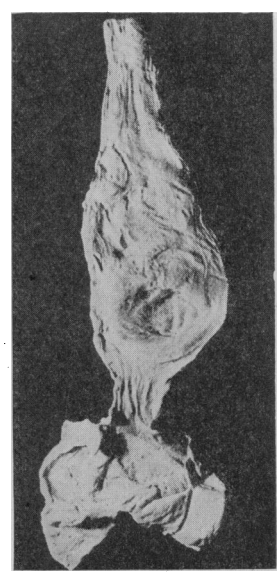

Fig. 8.—The same case. esophagus is opened, showing the dilatation and the thickness of the wall.

effervescing salt often overcomes the resistance. This is again studied under the screen. Inhalation of a vial of amyl-nitrite broken in a handkerchief accomplishes the same object. Several roentgenograms are then taken, in both anteroposterior and lateral positions, to check the fluoroscopic findings and again the roentgenologist and the surgeon go over the films together to avoid possible errors in the diagnosis.

Finally esophagoscopy is performed and the local disorders, such as ulcer, carcinoma, fissure, etc., are eliminated. Malignancy at the cardia, at times, is difficult to differentiate. In doubtful cases biopsy is done.

## TREATMENT.

The treatment of cardiospasm is essentially symptomatic. Shaw and Woo [22] classified the treatment according to the severity of the symptoms:

(*a*) Those of slight degree of obstruction require only careful dieting and attention to the general hygiene. The food should be nonirritating, highly nourishing in nature. This includes plenty of eggs, milk, cream, butter, well-cooked cereal, and fine vegetables. Meat should be given sparingly, if any, and then only scraped. It is much better to give a smaller quantity of food at frequent intervals.

(*b*) Those of more marked symptoms, but admitting a small-sized bougie, should have careful, systematic dilatation. This is by no means a new method. As early as in 1888 Russell [23] reported a sufficient series of cases to demonstrate the efficacy of this procedure.

For this purpose the Mosher bag is introduced under a fluoroscopic screen. When the tip drops into the hiatus the bag is gently passed an inch or two and a measured amount of air pressure is carefully exerted by inflating the bag. Excellent results are obtained by this method, and the patients may remain free from symptoms for weeks at a time when another such treatment is repeated.

(*c*) Finally those cases in which the obstruction is so complete as to prevent admission of even the smallest bougie should have gastrostomy, followed by some form of dilatation. Walton successfully treated sixteen such cases with only one death and that in a man sixty-six years of ago, who was already extremely emaciated, and died of heart failure seven days after operation.

Sedatives, which were much in vogue in the past, can no longer be considered efficacious. In view of the fact that a large number of these patients have a long-standing history, even dating back to childhood, every effort should be made for an early recognition of the presence of the condition, and proper and prompt treatment be instituted by the hands of an expert.

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## DISCUSSION

D. D. Comstock, M. D. (401 Bailey Street, Los Angeles).—There are many things about cardiospasm that would lead one to concur in the opinion expressed in Doctor Hara's paper that the background of this affliction is an inherited lack of balance in the vegetative nervous system, in which predisposing soil there has been cultivated an added factor of nerve stress and strain with nutritional deficiency. It seems apparent also in most cases that in the course of time a chronic inflammatory element is added. In one of our

patients this was so evident that hot and cold douching of the esophagus was resorted to with definite benefit. This was accomplished by passing a small stomach tube into the pouch and alternately filling and aspirating the contents by the use of a hard rubber syringe. By thus washing out the mucus and decomposing food, the passage of the silk thread was facilitated. We have observed patients in whom tenacious milk curds and other foods would be retained for hours or even days. This atonic and dilated state of the esophagus is doubtless as much a part of the affliction as the cardiospasm itself and not simply secondary. It therefore should receive therapeutic consideration from the start. In one of our patients the stricture became so pronounced that not even water would pass through and, of course, neither was the passage of the thread successful. But before resorting to a gastrotomy a very gentle but persevering effort was made to pass a small-sized piano-wire bougie direct. This was passed down to the stricture through a quite stiff stomach tube which was not fenestrated on the side. After a half-hour of teasing, the bougie was successfully passed and, of course, the rest was easy. It should be added that thorough lubrication of the inside of the stomach tube was necessary.

One having much of this work to do should learn to build his own dilating bags which, if designed somewhat after the lines of an hourglass, are more sure to be held firmly in the stricture during the process of dilatation. The suggestion that emphasis should be placed upon building up the general nerve and muscle tone and nutrition of the patient is important.

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KENNETH S. DAVIS, M. D. (Saint Vincent's Hospital, Los Angeles).—The chief roentgenographic characteristics of typical cardiospasm are the smooth, symmetrical, blunt or conical obstruction at or near the cardia and the secondary dilatation of the esophagus above. This dilatation is usually marked; in some instances involving the entire esophagus. In cases of long duration the esophagus is usually somewhat elongated and may become either kinked or S-shaped. Thus we find the spindle-shaped, the pear-shaped, and the S-shaped types of dilated esophagus as described in the article under discussion.

Reversed peristalsis may be seen during the fluoroscopic examination; this finding, however, is very rare. Quite commonly the dilated esophagus is found to be filled with fluid through which the opaque barium slowly sinks in blobs.

From the roentgenographic viewpoint cardiospasm is to be differentiated chiefly from carcinoma and benign organic stricture. Carcinoma seldom ends exactly at the hiatus and there is usually noted an irregularity of the lower end of the barium shadow, whereas in cardiospasm the shadow is symmetrical and smooth. Furthermore carcinoma rarely causes the extreme dilatation of the esophagus so commonly seen in cardiospasm. Posttraumatic strictures generally occur higher in the esophagus than the cardia, and one can reasonably expect some irregularity at the site of the lesion. As Hara has already pointed out, an inquiry should be made as to the swallowing of corrosives in all suspected cases.

In view of the fact that filling defects at the site of the obstruction do not always signify organic lesion, and smooth, symmetrical regularity always indicate cardiospasm, great care should be taken to carefully coördinate the clinical and the roentgenographic findings. Esophagoscopy should be done in any case with indefinite clinical and roentgenographic findings.

The author states that gastrostomy should be performed in cases of cardiospasm in which bougies cannot be passed. In a large series of cases Vinson has seldom found it necessary to resort to this procedure, due to his success in passing bougies. He does this by first having the patient swallow a thread which, when anchored, serves as a guide for the bougie. By tensing the thread the bougie can be directed exactly to the constricted area which is then dilated. This method also eliminates danger of perforation.

For the fluoroscopic examination of the esophagus a special barium acacia mixture is commonly used. This mixture is exceedingly viscid and descends the esophagus slowly with a tendency to coat its walls rather than fill the lumen. In this manner filling defects are brought into plain view which ordinarily would be missed.

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# ACTINOMYCOSIS*

## REPORT OF CASES

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AND
PAULA SCHOENHOLZ, M. A.
*San Francisco*

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ACTINOMYCOSIS is a well-known clinical entity and has been carefully described in all its details by many observers. The purpose of this report is to summarize some advances which have contributed to a better understanding of the condition, and have led to a more rational plan of treatment. The discussion is limited to actinomycosis of the jaw.

### EARLY STUDIES ON ACTINOMYCOSIS

The view that actinomycosis is contracted by the introduction of such substances as infected grain, straw, and grasses into the oral cavity dates back to the work of Bostroem [1] in 1890. He had isolated a streptothrix from human cases of actinomycosis which was apparently identical with an organism found widespread in nature upon grasses and grains. This theory has been handed down in textbooks and in the lecture room since that time. Homer Wright [2] showed that the true *Actinomyces bovis,* first described by Israel and Wolff [3] in 1878, is not found outside of the animal body and that it has quite different cultural characteristics from the streptothrix of Bostroem. *Actinomyces bovis* is a compulsory anaërobe, difficult to grow, and grown best at body temperature. On the other hand Bostroem's organism grows luxuriantly on ordinary culture media, at room temperature, and is an aërobe.

F. T. Lord[4] in 1910 reported that he had isolated *Actinomyces bovis* from carious teeth and cryptic tonsils in patients who had no evidence of actinomycosis. This work seems to indicate that *Actinomyces bovis* may be a saprophyte which exists in the normal mouth and gastro-intestinal tract.

The source of infection in many cases of actinomycosis has been quite obscure, no history of contact with infected cattle being obtained. Many patients deny that they chew straw or grass or that they have been connected with rural life in any way. From a clinical standpoint it would seem more probable that the organisms exist as

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